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OCCUPATIONAL VINYL CHLORIDE INTOXICATION

[O professional'noy intoksikatsii vinilkhloridom]

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OCCUPATIONAL VINYL CHLORIDE INTOXICATION

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Cases of acute vinyl chloride intoxication in workers are extremely rare and are usually due to massive exposure to the substance during work in closed areas and during accidents. Under the indicated conditions even short-term breathing of vinyl chloride can cause sudden loss of consciousness and a state of narcosis. In those cases when the patient is not immediately removed from the gas-filled room, a coma may develop that is lethal as the result of respiratory arrest. With timely rendering of first aid, patients soon emerge from the comatose condition, but consciousness may remain clouded for a short time. Over the next few days, headache, dizziness, general weakness and an inclination to bradycardia and arterial hypotony are observed. The patient practically recovers in 5-7 days.

Only mild forms of acute vinyl chloride intoxication may be observed under modern industrial conditions. Clinically, these forms are of the prenarcosis state type. Upon withdrawing to fresh air, the signs of intoxication are rapidly eliminated. Chronic occupational vinyl chloride intoxication develops after exposure over a period ranging from 4-5 months to several years. In the initial period of chronic intoxication, the clinical picture is distinguished by polymorphism, significant variability of the course, an inclination to vegetative paroxysms with disorders of the vegetative-vascular, thermoregulatory, neurotrophic and neuro-endocrine regulation, which indicates dysfunction of the brain stem-hypothalamic structures. A slight increase in size of the liver may be simultaneously observed without clearly defined disorders of hepatic functions, with urticarious eruptions on the skin and a tendency to anemization with mild hemolytic symptoms.

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The neurologic symptoms of the intoxication also vary depending on the nature of development of the process. Thus, in persons subjected to repeated mild acute vinyl chloride intoxications against a background of toxic exposure, one primarily notes pronounced disorders of the neuroregulatory processes accompanied by vegetative crises and vascular paroxyisms. Moreover, osteotrophic disorders manifested in osteolysis of the toes are characteristic.

Disruption of temperature regulation appears in seizures of chills, general chilliness, a reduction in skin temperature and an inclination to subfebrility in the evening (37.1 - 37.8°). The neuro-endocrine disorders appear in a significant loss of weight (4-15 kg), loss of sexual potency in men and a slight increase or decrease in thyroid function without clearly defined disorders of the basal metabolism. Additionally, patients are observed to have other clinical symptoms which confirm the involvement of the brain-stem regions in the process. We observed sleep disorders, pronounced asthenization with seizures of severe adynamia, microsymptoms of disruption of the craniospinal innervation (oculomotor, trigeminal) and of the motor and sensory spheres.

Chronic intoxication develops over 3-5 years in people who have not suffered repeated acute poisonings. In this case, the developing asthenic symptoms gradually emerge in foremost. The osteotrophic disorders in these cases are manifested in foci of osteoporosis and osteosclerosis, predominantly in the fingers.

In the I stage, the process is not persistent and a tendency to remission is detected soon after terminating contact with the toxic agent. Vegetative-sensory polyneuritis develops in the II stage of chronic intoxication against the background of the syndrome described above. Among the surface types of sensitivity, pain sensitivity is the most clearly defined, and is lowered. Motor disorders are not characteristic for this stage of intoxication.

The vegetative-vascular paroxyms acquire a biphase character: vascular spasms alternate with a stage of atony. A spastico-atonic condition of the

capillaries with the appearance of intracapillary and intercapillary anastomosis is determined capillaroscopically. Disorders of the cardiac activity (pronounced sinus arythmia, extrasystole) often appear in this stage of intoxication, as do pains in the region of the heart. In this case one can detect deficiency of the coronary circulation and disruption of nutrition of the heart muscle on the electrocardiogram. Disruption of the adaptation of the organism to environmental changes is clearly pronounced.

General asthenization increases in the III stage. A depressed mood develops and in certain patients elementary hallucinations, insomnia, and pronounced oculomotor disorders appear: periodic double vision, forceful rolling of the eyes. Rapid movements of the eyeballs often produce visually induced convulsions and an unpleasant sense of nausea and dizziness. In this case the face and chest intensively redden, the pulse and respiration increase in frequency, or rarely, on the other hand, slow. Crying and occasionally powerful weeping simultaneously appear. Disorders of innervation of the trigeminal nerve may spread not only to the sensory portion but also to the motor one. In individual cases, signs of injury to the nuclei of the facial and sublingual nerves appear. The tendinous reflexes are elevated. Hence, in the III stage of intoxication the cerebral disorders are linked to primary injury of the brain stem region. Toxic polyneuritis in this stage is characterized not only by vegetative-sensory disorders, but also by motor disorders, although the latter usually do not reach the stage of paresis. The vegetative-vascular disorders are characterized predominantly by symptoms of vasoparesis.

An inclination to arterial hypertension is noted (occasionally to high figures), the pulse is often arythmic with an inclination to increase up to 120 beats per minute. Disorders of rhythm of the cardiac activity and dystrophic changes in the myocardium are electrocardiographically detected. The examination of the gastrointestinal tract in many cases shows reduced acidity of the gastric juice up to total achilia and also shows atony of the stomach. Occasionally, limited and diffused thickening of the skin and subcutaneous tissue of the sclerodermic type are noted; a tendency toward erythrocytosis is noted with the appearance of the large forms of erythrocytes; leucocytosis

appears as the result of an increase in eosinophils and monocytes and there is a reduction in the content of blood platelets (Ye.P, Rumyantseva).

Observation shows that only symptoms of the I stage of intoxication are fully reversible. Beginning with the II stage, most of the symptoms of intoxication responds poorly to treatment and in a number of cases there is an inclination to progression of the process, notwithstanding determination of contact with the toxic substance and intensive treatment.

The cited data show the pressing need for recognizing the initial forms of vinyl chloride intoxication and for timely, effective occupational assignment of these people.